

SEVERE TRICUSPID STENOSIS REVEALED AFTER AORTIC VALVOTOMY

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Surgical treatment of a rheumatic valvular lesion may exaggerate the physical signs or lead to the recognition for the first time of another valvular lesion. This is seen in combined lesions of the aortic and mitral valves and less frequently of the mitral and tricuspid valve. Tricuspid stenosis (Pantridge and Marshall, 1957) and tricuspid incompetence (Mounsey, 1959) have been revealed after mitral valvotomy but not after aortic valvotomy. This is probably due to the fact that rheumatic tricuspid disease is nearly always associated with mitral disease, less commonly with mitral and aortic valve disease and rarely with aortic disease alone (Gibson and Wood, 1955; Goodwin *et al.*, 1957; Killip and Lukas, 1958; Perloff and Harvey, 1960). The following case report is of a young patient in whom tricuspid stenosis was revealed after aortic valvotomy. The tricuspid lesion accounted for unexpected failure to improve after a technically successful aortic valvotomy but its surgical correction at a second operation led to marked clinical improvement.

Case Report

Elizabeth McL., aged 25, was first seen in April 1957. She had a history of tonsillectomy when 7 and of rheumatic fever and chorea when 8 years old. For about 2 years she had complained of increasing tiredness and breathlessness on exertion, palpitation and occasional præcordial pain or giddiness on unaccustomed exercise. She could only climb 8 steps without having to pause for breath. She was a well nourished, healthy-looking young woman. Her arterial pulses were small and sustained and the heart rate 50 per minute. Examination of the heart, which was not clinically enlarged, suggested left ventricular hypertrophy and the dominant physical sign was a harsh basal systolic murmur widely conducted, felt as a thrill at the aortic area and accompanied by a soft aortic diastolic murmur. Venous pulsation was noted in the root of her neck to a height of about 1 cm. above the sternal angle but a tricuspid lesion was not suspected at this time. Her brachial arterial pressure was 130/100 mm. Hg. Radiological examination showed left ventricular enlargement and a prominent ascending aorta; there was no left atrial enlargement and no valvular calcification (Fig. 1). She was considered to have severe rheumatic aortic stenosis without significant incompetence and in view of her age and symptoms operation was advised.

A transventricular aortic valvotomy was carried out on 7/6/57 with satisfactory splitting of the stenosed valve. The post-operative period was uneventful and her blood pressure was now about 120/75 mm. Hg. Her subsequent progress was disappointing, there being little or no symptomatic improvement and she complained bitterly of "being unable to get a move on." Soon after her operation she became aware of a new symptom—a fluttering sensation in her neck, and prominent pulsations were noticed there both by herself and her relatives. When seen again in January 1958 these were obviously giant "a" waves and, on careful auscultation, a tricuspid pre-systolic murmur and opening snap were heard (Fig. 2A). Furthermore, serial X-rays showed that the right atrium had become prominent since her operation (Fig. 1) and examination of serial electrocardiograms showed that there had previously been evidence of right atrial hypertrophy. Cardiac catheterization on 2/5/60 confirmed the clinical diagnosis of severe tricuspid stenosis (Fig. 2B). There was a low resting cardiac output (2.8 litres per minute), normal pressures in the wedged pulmonary artery position (-1.5 mm. Hg mean), pulmonary artery (29/10 mm. Hg) and right ventricle (29/ -4 mm. Hg) and raised right atrial pressure (22/0 mm. Hg) with giant "a" waves and an atrio-ventricular pressure gradient.

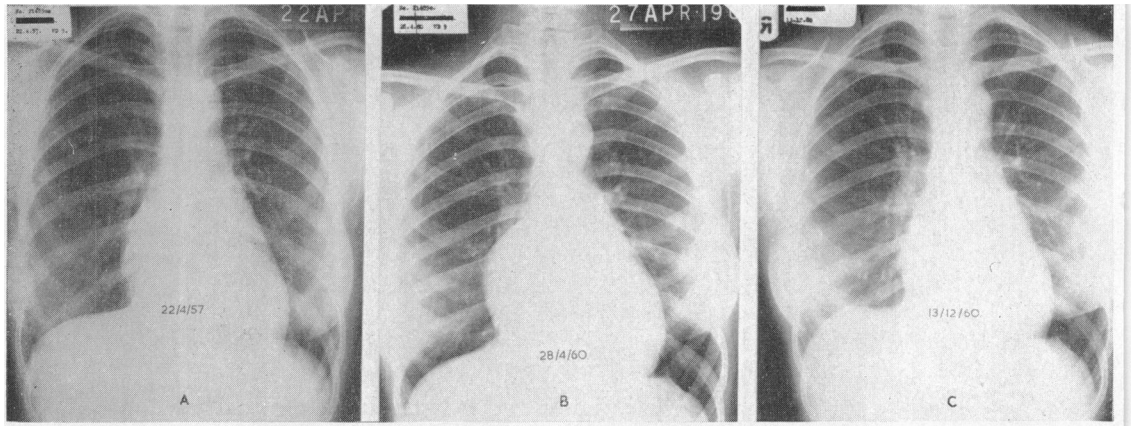


FIG. 1.—Serial chest X-rays: (A) 22/4/57, before aortic valvotomy; (B) 28/4/60, before tricuspid valvotomy, and (C) 13/12/60, after tricuspid valvotomy. Note the increase in size of the right atrium in (B) following aortic valvotomy and its regression in size after tricuspid valvotomy.

Because of her continued severe handicap a second operation was advised and a tricuspid valvotomy was carried out on 19/7/60; the valve was found to be less than 1 cm. in diameter, the cusps were mobile and there was no regurgitant jet. The commissures were split with difficulty using digital pressure and an aperture, 3 cm. in diameter, was obtained.

Again she had a satisfactory post-operative recovery and this time showed an immediate improvement in exercise tolerance. She could now climb a long staircase without stopping and was able to hurry on the flat in a quite unaccustomed way. She was progressing well at home until 28/8/60 when she developed palpitation associated with nausea and breathlessness and on readmission to hospital was found to be having paroxysms of atrial fibrillation, atrial tachycardia and frequent runs of atrial ectopic beats. Treatment proved difficult, both quinidine and digitalis being required to suppress the ectopic rhythm. It was feared that there had been a reactivation of rheumatism but no proof of this was forthcoming.

When last seen in December 1960 she was very well and much less handicapped than she could ever remember. Though the signs of aortic stenosis remain it would now be difficult to diagnose tricuspid stenosis. Her blood pressure remains 120/75 mm. Hg, her electrocardiogram shows regression of the high voltage P waves, and radiologically the right atrium is much less prominent (Fig. 1).

Discussion

This patient had rheumatic aortic and tricuspid stenosis. The absence of mitral murmurs, left atrial enlargement or elevation of the pulmonary artery "wedge" pressure excluded significant involvement of the mitral valve. When she was first seen only the aortic lesion was diagnosed and it was disappointing when a technically satisfactory aortic valvotomy did not lead to any lessening of her symptoms. The signs of tricuspid stenosis, which were probably minimal and overlooked before the aortic valvotomy, became very obvious after it, and this lesion appeared now to be the main cause of continued disability. Tricuspid valvotomy led to marked clinical improvement. The mechanism of the unmasking of tricuspid stenosis by aortic valvotomy is not clear. Mounsey (1959) also had difficulty in explaining the unmasking of rheumatic tricuspid incompetence after technically successful mitral valvotomy but he thought that increased stroke output might be one important factor. In our patient it may be that increased stroke volume or a modest increase in cardiac output was of importance in unmasking the tricuspid stenosis. There is thus an obvious need for further investigation of right heart function after mitral or aortic valvotomy in patients with rheumatic heart disease.

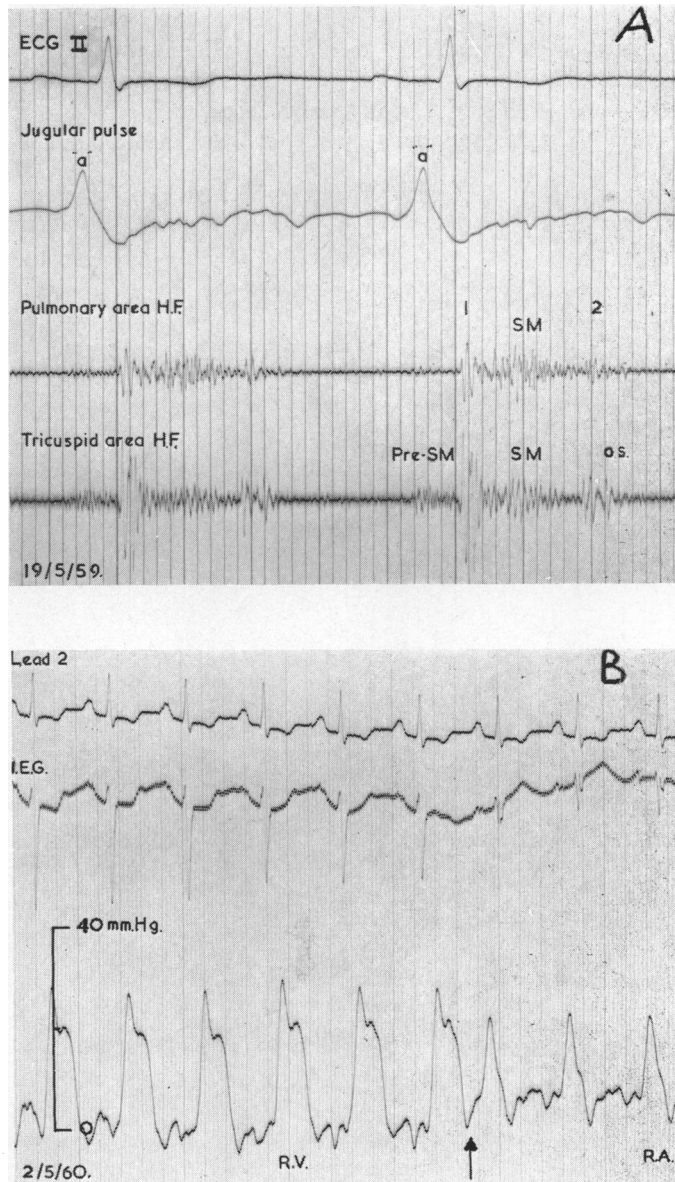


FIG. 2.—(A) Phonocardiograms and jugular phlebogram made two years after aortic valvotomy showing the relationship between the giant “a” waves in the jugular venous pulse and the tricuspid presystolic murmur. This murmur increased in intensity throughout inspiration and during this phase of respiration an opening snap could be heard and recorded. Its timing in relation to the second heart sound varied from 0.06 seconds in early inspiration to 0.10 seconds at the height of inspiration. The widely conducted aortic systolic murmur, though still present, is of greatly reduced amplitude. (B) A tracing made during cardiac catheterization, as an electrode catheter was withdrawn from the right ventricle (R.V.) through the stenosed tricuspid valve into the right atrium (R.A.) showing the giant “a” waves in the atrium and the considerable pressure gradient between the two chambers. The change in pressure pulse, shown by the arrow, takes place in mid-diastole, the change in the intracardiac electrogram (I.E.G.) pinpointing the site of the tricuspid valve in the record.

HF=High frequency.
OS=Opening snap.

Pre-SM=Presystolic murmur.
1=1st heart sound.

SM=Systolic murmur.
2=2nd heart sound.

Summary

The case of a young woman who failed to show clinical improvement after valvotomy for aortic stenosis is described. In the post-operative period signs of gross tricuspid stenosis became prominent for the first time, possibly due to altered hæmodynamics following relief of the aortic valve obstruction. Tricuspid valvotomy subsequently led to marked clinical improvement.

We are grateful to Professor I. G. W. Hill for permission to publish this case and to Professor D. M. Douglas for the operative findings.

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